LCook 1/11/08

PALM INTRANET

Day : Friday
Date: 1/11/2008
Time: 18:47:06

Biotech Query for 10/629975

Title: **METHOD FOR DIFFERENTIATING IRRITABLE BOWEL SYNDROME FROM INFLAMMATORY BOWEL DISEASE (IBD) AND FOR MONITORING PERSONS WITH IBD USING TOTAL ENDOGENOUS LACTOFERRIN AS A MARKER**

Inventor: **BOONE, JAMES**

Location:

Location Date: Group Art Unit: **1641**

Status: **71/RESPONSE TO NON-FINAL OFFICE ACTION ENTERED AND FORWARDED TO EXAMINER**

Barcode: Filing or 371(c) Date: **07/30/2003**

Num	Date	Code	Contents Description
NO BIOTECH DATA			

Search for Biotech Info: **Application#** [ ] Search

PCT / [ ] / [ ] Search

Bar Code # [ ] Search

To go back, right click here and select Back. To go forward, right click here and select Forward. To refresh, right click here and select Refresh.
Back to OASIS | Home page

L/Cook
1/11/08

PALM Intranet

Application Number Submit

IDS Flag Clearance for Application 10629975

IDS Information

Content	Mailroom Date	Entry Number	IDS Review	Last Modified	Reviewer
Update					

10/629,975
Search
L4Cook 1/11/08

d his

(FILE 'HOME' ENTERED AT 18:52:23 ON 11 JAN 2008)

FILE 'BIOSIS, CAPLUS, EMBASE, JAPIO, MEDLINE' ENTERED AT 18:53:21 ON 11 JAN 2008

L1	24620	S	CALCITRIOL AND HUMAN
L2	21	S	L1 AND IBD
L3	16403	S	(DEXTRAN SULFATE)
L4	1	S	L3 AND L1
L5	451	S	L3 AND IBD
L6	171	S	L5 AND HUMAN?
L7	16	S	L6 AND PD<2001
L8	9		DUPLICATE REMOVE L7 (7 DUPLICATES REMOVED)

=>

ANSWER 8 OF 9 CAPLUS COPYRIGHT 2008 ACS on STN
AN 1996:589875 CAPLUS
DN 125:298289
ED Entered STN: 03 Oct 1996
TI Development of an experimental colitis in animal models
AU Kitano, Atsuo
CS Juso Citizens' Hosp., Osaka, 532, Japan
SO Igaku no Ayumi (1996), 178(9), 644-648
CODEN: IGAYAY; ISSN: 0039-2359
PB Ishiyaku
DT Journal; General Review
LA Japanese
CC 14-0 (Mammalian Pathological Biochemistry)
AB A review, with 31 refs., on the animal models of idiopathic inflammatory bowel disease (IBD) compared with human ulcerative colitis (UC) and Crohn's disease (CD) on the lymphocytes and inflammatory mediators. Described are spontaneous models of Cotton-top tamarin and juvenile rhesus macaques, and models by chemical substances as acetic acid, EtOH, sulfated polysaccharides as carrageenan and dextran sulfate sodium (DSS). The models are also established using specimens of UC patients by manipulating animal immune system as B cell models and T cell models.
ST review colitis animal model
IT Intestine, disease
(colitis, development of exptl. colitis in animal models)

ANSWER 2 OF 9 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2000:511181 BIOSIS
DN PREV200000511181
TI Novel therapies help dissect the inflammatory pathways of dextran sulfate (DSS)-induced mouse colitis: Application for the treatment of human inflammatory bowel diseases.
AU Flanigan, A. [Reprint author]; Murthy, S. [Reprint author]
CS MCP Hahnemann University, Philadelphia, PA, 19102, USA
SO Inflammation Research, (August, 2000) Vol. 49, No. Supplement 2, pp. S94. print.
Meeting Info.: 10th National Conference of the Inflammation Research Association. Hot Springs, Virginia, USA. September 24-28, 2000.
ISSN: 1023-3830.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 22 Nov 2000
Last Updated on STN: 11 Jan 2002
CC Digestive system - Physiology and biochemistry 14004
General biology - Symposia, transactions and proceedings 00520
Biochemistry studies - Carbohydrates 10068
Pathology - Therapy 12512
Digestive system - Pathology 14006
Pharmacology - General 22002
IT Major Concepts
Digestive System (Ingestion and Assimilation); Pharmacology
IT Diseases
colitis: digestive system disease
Colitis (MeSH)
IT Diseases
inflammatory bowel disease: digestive system disease, IBD
Inflammatory Bowel Diseases (MeSH)
IT Chemicals & Biochemicals
antiinflammatory drugs; dextran sulfate
IT Miscellaneous Descriptors
Meeting Abstract
ORGN Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
mouse
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates
RN 9042-14-2 (dextran sulfate)

10/629,975
Search
L/Cook 1/11/08

```
d his

     (FILE 'HOME' ENTERED AT 17:46:46 ON 11 JAN 2008)

     FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, JAPIO' ENTERED AT 17:47:13 ON 11
     JAN 2008
L1           132 S (ANIMAL MODEL) AND (HUMAN IBD)
L2            76 DUPLICATE REMOVE L1 (56 DUPLICATES REMOVED)
L3             0 S L2 AND CALCITRIOL?
L4            47 S L2 AND MOUSE?
L5            15 S L2 AND DEXTRAN
L6            12 S L5 AND SULFATE?
L7            11 S L6 AND L4

=>
```

ANSWER 8 OF 11 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN

AN 2005457111 EMBASE

TI Interleukin-1β targets interleukin-6 in progressing dextran sulfate sodium-induced experimental colitis.

AU Ki H.K.; Murakami A.; Hayashi R.; Ohigashi H.

CS H. Ohigashi, Division of Food Science and Biotechnology, Graduate School of Agriculture, Kyoto University, Kyoto 606-8502, Japan. ohigashi@kais.kyoto-u.ac.jp

SO Biochemical and Biophysical Research Communications, (18 Nov 2005) Vol. 337, No. 2, pp. 647-654.
Refs: 50
ISSN: 0006-291X E-ISSN: 1090-2104 CODEN: BBRCA9

PUI S 0006-291X(05)02131-5

CY United States

DT Journal; Article

FS 026 Immunology, Serology and Transplantation
029 Clinical and Experimental Biochemistry
048 Gastroenterology
005 General Pathology and Pathological Anatomy

LA English

SL English

ED Entered STN: 27 Oct 2005
Last Updated on STN: 27 Oct 2005

AB Inflammatory bowel disease (IBD) is an immunologically mediated disorder that is characterized by chronic, relapsing, and inflammatory responses. Dextran sulfate sodium (DSS)-induced experimental colitis in mice has been recognized as a useful model for human IBD and interleukin (IL)-1β is a key cytokine in the onset of IBD. The purpose of the present study was to clarify which pro-inflammatory mediators are targeted by IL-1β in mice with DSS-induced colitis. First, we found that DSS markedly induced IL-1β production in both dose- and time-dependent manners ($P < 0.05$ and $P < 0.01$, respectively) in murine peritoneal macrophages (pMθ), while that of tumor necrosis factor-α was insignificant. Further, the expressions of mRNA and protein for IL-1β were increased in colonic mucosa and pMθ from mice that received drinking water containing 5% DSS for 7 days ($P < 0.01$, each). In addition, the expressions of IL-6, granulocyte macrophage-colony stimulating factor, inducible nitric oxide synthase, and cyclooxygenase-2 mRNA were also time dependently increased ($P < 0.01$, each). Furthermore, administration of rIL-1β (10 μg/kg, i.p.) significantly induced the expressions of IL-1β and IL-6 mRNA in colonic mucosa from non-treated mice ($P < 0.01$). Anti-mIL-1β antibody treatments (50 μg/kg, i.p.) attenuated DSS-induced body weight reduction and shortening of the colorectum ($P < 0.05$, each), and abrogated the expressions of IL-1β and IL-6 mRNA in colonic mucosa ($P < 0.01$, each). Our results evidently support the previous findings that IL-1β is involved in the development of DSS-induced experimental colitis in mice, and strongly suggest that IL-1β targets itself and IL-6 for progressing colonic inflammation.

CT Medical Descriptors:
animal cell
animal experiment
animal model
animal tissue
article
*colitis
colon mucosa
controlled study
dose response
female
inflammation
mouse

nonhuman
peritoneum macrophage
priority journal
protein expression
weight reduction

CT Drug Descriptors:
cyclooxygenase 2
cytokine antibody
*dextran sulfate
drinking water
granulocyte macrophage colony stimulating factor
inducible nitric oxide synthase
*interleukin 1beta
*interleukin 6
messenger RNA: EC, endogenous compound
recombinant interleukin 1beta
tumor necrosis factor alpha

RN (dextran sulfate) 9011-18-1, 9042-14-2; (inducible nitric oxide synthase) 501433-35-8

ANSWER 10 OF 11 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN

AN 2003512020 EMBASE

TI Increased disease activity in eNOS-deficient mice in experimental colitis.

AU Sasaki M.; Bharwani S.; Jordan P.; Elrod J.W.; Grisham M.B.; Jackson T.H.; Lefer D.J.; Alexander J.S.

CS Dr. J.S. Alexander, Dept. of Molec. and Cell. Physiology, LSU Health Sciences Center, 1501 Kings Highway, Shreveport, LA 71130-3932, United States. jalexa@lsuhsc.edu

SO Free Radical Biology and Medicine, (15 Dec 2003) Vol. 35, No. 12, pp. 1679-1687.
Refs: 54
ISSN: 0891-5849 CODEN: FRBMEH

CY United States

DT Journal; Article

FS 029 Clinical and Experimental Biochemistry
048 Gastroenterology
005 General Pathology and Pathological Anatomy

LA English

SL English

ED Entered STN: 22 Jan 2004
Last Updated on STN: 22 Jan 2004

AB Oral dextran sodium sulfate (DSS, 3%) produces experimental colitis with many features of human inflammatory bowel disease (IBD), (leukocyte extravasation, cachexia, and histopathology). Previous studies suggest that the inducible nitric oxide synthase (iNOS) in blood cells or in the endothelium contribute to this injury. However, until now no study has been performed to directly evaluate the role of endothelial nitric oxide synthase (eNOS) in IBD. We compared disease activity in wild-type (eNOS(+/+)) and eNOS-deficient (eNOS(-/-)) mice in the DSS model of colitis. Administration of DSS induced weight loss, stool blood, and overt histopathology in both mouse strains. Disease activity was dramatically increased in eNOS(-/-) mice compared to wild types. Histologically, eNOS-deficient mice had greater leukocyte infiltration, gut injury, and expressed higher levels of the mucosal addressin, MAdCAM-1. These results demonstrate that eNOS plays an important role in limiting injury to the intestine during experimental colitis and altered eNOS content and/or activity may contribute to human IBD. .COPYRGT. 2003 Elsevier Inc.

CT Medical Descriptors:
animal experiment
  animal model
animal tissue
article
blood cell
cachexia
*colitis: ET, etiology
controlled study
*disease activity
*disease course
endothelium
enteritis
enzyme activity
experimental model
histopathology
intestine injury
  knockout mouse
leukocyte
lymphocytic infiltration
  mouse
nonhuman
occult blood
priority journal
weight reduction

wild type

CT Drug Descriptors:
addressin: EC, endogenous compound
dextran derivative
dextran sulfate
mucosal addressin cell adhesion molecule 1: EC, endogenous compound
*nitric oxide synthase: EC, endogenous compound

RN (dextran sulfate) 9011-18-1, 9042-14-2; (mucosal addressin cell adhesion molecule 1) 181789-23-1; (nitric oxide synthase) 125978-95-2